



Letter to the Editor

Electrocardiographic changes in attempted-hanging



Sir,

The case report by Aslam and Maurya is indeed interesting.¹ However, we would like to further highlight few aspects of this paper. Apart from the involvement of the neurocardiac axis, two other mechanisms that might have contributed to myocardial and electrocardiographic (ECG) changes are hypoxia mediated catecholamine surge and hyperventilation.² Attempted-hanging affects the heart that includes morphological change and ischemia, which may precipitate arrhythmias.³ Clinically, the chronology of ECG changes observed in this case makes us to consider the possibility of Takatsubo cardiomyopathy.⁴ Moreover, the presence of prominent U waves has been proposed as an additional ECG finding to support the diagnosis of Takatsubo cardiomyopathy.⁵

Excessive catecholamine stimulation may result in microvascular coronary spasm or endothelial dysfunction, and lead to direct catecholamine-mediated myocyte injury, which manifests as contraction band necrosis. This is histopathologically differentiated from coagulation necrosis, seen in myocardial infarction resulting from coronary artery occlusion.⁶ The presence of microvascular damage seems to be the most powerful determinant of persistent ST-segment elevation as reported in this case.

Ultimately the clinicians, if they happen to observe such ECG changes in an unconscious or a restless patient, may consider various conditions (non-ischemic cardiac causes) that can be associated with ST-segment elevation and rule out one from another based on clinical and laboratory data. The awareness of such entities and being familiar with the electrocardiographic features that distinguish them from infarction help an astute physician to make a reasonable diagnosis and find out the probable cause at the bedside, and provide appropriate management.⁷

Conflict of interest

None.

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